

AD-A267 441



20

# INSTITUTE FOR BRAIN AND NEURAL SYSTEMS

June 30, 1993

Dr. Joel L. Davis, Program Manager  
Office of Naval Research  
Code 1142 BI  
800 N. Quincy Street  
Arlington, Virginia 22217-5000

DTIC  
ELECTE  
AUG 04 1993  
S A D

Re: Progress Report N00014-91-J-1316

Dear Dr. Davis:

This is written to provide a semi-annual progress report for the contract N00014-91-J-1316 entitled "Theoretical and Experimental Research into Biological Mechanisms Underlying Learning and Memory." The major goal of our research is to elucidate the biological mechanisms that underlie learning and memory: to find principles of organization that can account both for experimental data on the cellular level and, when applied to large numbers of neurons that receive sensory and/or interneuronal information, for various higher level system properties.

Among our detailed objectives are the following: to clarify the dependence of learning on synaptic modification, to elucidate the principles that govern synapse formation or modification, to use principles of organization that can account for observations on a cellular level to construct neural-like systems that can learn, associate and reproduce such higher level cognitive acts as abstraction and computation.

The approaches employed to achieve these objectives include both theory and experiment. Theoretical and experimental consequences of the hypothesis that synapse modification is dependent on local information (in visual cortex, dominated by the inputs from the eyes with specific visual information) in accordance with theoretical ideas we have developed, as well as by global instructions affecting large numbers of synapses and coming perhaps from modulatory transmitters such as norepinephrine, have been tested. In addition, various

93 8 3 108<sup>1</sup>

~~93 7 06 028~~

93-17410

348 Y

principles that appear to be operating on the cellular level have been used to construct models of higher level functions.

One of our key objectives is to produce real interaction between theory and experiment. The means for achieving this has been a continuing dialogue between experimentalists and theoreticians that has produced a genuine collegial relationship in which experts in very different disciplines can understand each other's language.

## 1 Simulations using natural inputs

A key simplification used up to now in simulations and analysis of the evolution of BCM neurons has been the visual environment. In the past contract period we have begun and investigation of the validity of this rearing environment model used in the CBC simulations of visual deprivation experiments was tested by using a more realistic model of visual experience. Natural images preprocessed by a retinal filter were used to generate input to a single cell model of synaptic plasticity in visual cortex. The simulations of normal rearing, monocular deprivation and reverse suture using these realistic inputs produced similar results as the CBC simulations which used abstract one dimensional inputs.

These simulations used a model of the kitten visual system from the retina to primary visual cortex. A single neuron represented the cortex, and the BCM theory was used to model its synaptic plasticity. Circular regions from the left and the right retinas covering the same visual space, were used to generate input to the single BCM neuron. The lateral geniculate nucleus (LGN) was assumed to simply relay the signal generated by the retina to the visual cortex.

Each retina included an array of ganglion cells spaced one unit apart, and an array of receptors which were also spaced one unit apart. Only ganglion cells, whose receptive-field midpoints fell within a circular visual area with a radius of five units were included in the model. Each ganglion cell had an antagonistic center-surround receptive field which approximated a difference of two Gaussians. The standard deviation of the center Gaussian was 1 unit, and the standard deviation of the surround Gaussian was 3 units. This created a receptive field center with a radius of 2.22 units. The receptive field of each ganglion cell was balanced so that uniform illumination of any intensity resulted in spontaneous activity.

The visual environment of the model consisted of eight gray scale images with dimensions 150X150 pixels. For each cycle of the simulation, the activity of the receptors in the retina was determined by randomly picking one of the eight images, and randomly shifting the image on the models retina. The shift was restricted so that none of the ganglion cell receptive field centers fell within five units of the image border. The activity of each receptor in the model was determined by the intensity of a pixel in the image. This method generated a very large training set because of the many unique shifts which were possible. The maximum ganglion cell activity generated by the patterned input was 1.57, and the ganglion cell activity generated by a sutured eye was simply noise uniformly distributed in the interval [0.0, 0.8).

A selected times during the simulations, spots of light were used to characterize the receptive field of the BCM neuron through the left and right eyes. Two dimensional maps of the

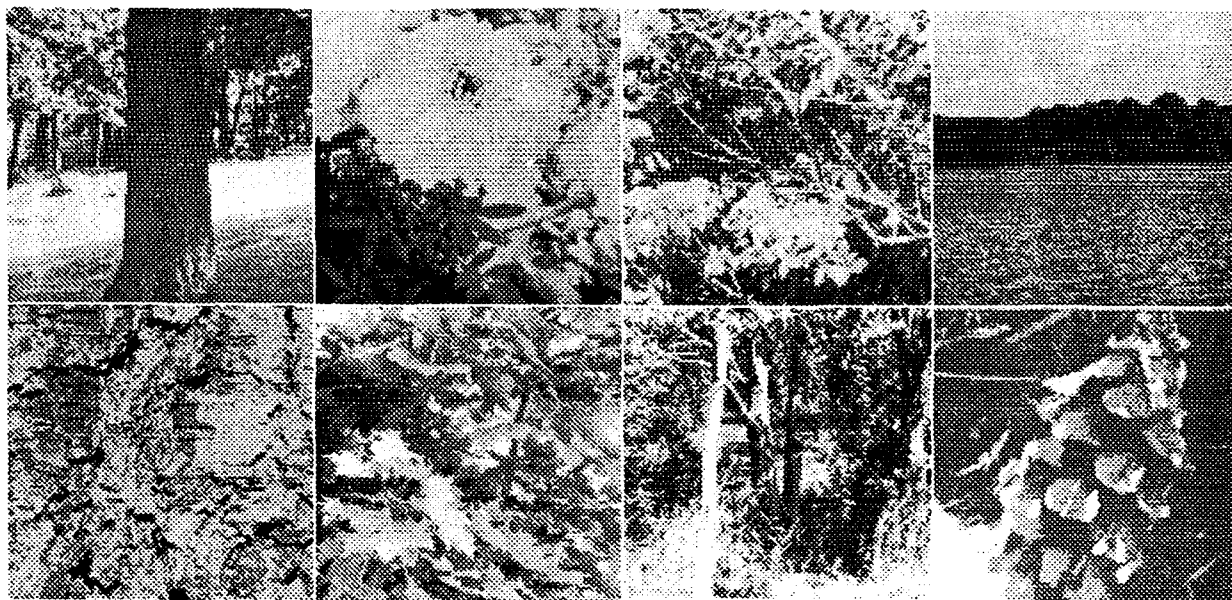


Figure 1: Natural images used for training the model.

receptive field were generated by "shining" small spots of light at many location on a retina and recording the BCM activity generated for each spot. This is similar to the process used by Palmer and Jones to generate two-dimensional receptive field profiles of simple cells in cat striate cortex (Jones and Palmer, 1987). The maximums of the left eye map and the right eye map were used to determine the binocularity of the BCM neuron.

Figures 3, 5 and 7 show the results from simulations of normal rearing, monocular deprivation and reverse suture. They can be compared to the results of the CBC simulations shown in figures 2, 4 and 6. The scale of the horizontal axis in these two sets of figures is different because the simulations using the natural input required many more training iterations for the BCM neuron to become selective. This can be accounted for by the additional complexity introduced by the realistic input. As in the CBC simulations, normal rearing produced a binocular neuron which was equally driven through the left and the right eyes. The two-dimensional maps of the BCM neurons receptive field show how it also develops selectivity to the orientation of a stimulus.

In both simulations of monocular deprivation, the sutured eye disconnects from the BCM neuron, and in both simulations of reverse suture the newly closed eye disconnects from the BCM neuron before the newly open eye reconnects. These results suggest that the original abstract patterns distorted by noise were an adequate model of visual experience for the simulations of these visual deprivation experiments.

P. <i>parthi</i>	
DATE	
A. <i>...</i>	
Dist	A. <i>...</i>
A-1	

## 2 Localized Principal Components of Natural Images - an Analytic Solution

It has been proven that a neuron with Hebbian learning rule plus a proper decay term can perform a principal component extraction. Furthermore, a neural network with proper lateral inhibition can perform the extraction of several principal components simultaneously. The computational importance of principal components is that they are the optimal linear projections for minimizing the mean squared reconstruction error.

Since the principal components of a set of inputs depend only on their covariance matrix, it is reasonable that given this matrix, they can be calculated analytically.

We believe that it is reasonable to model postnatal development with an environment composed of natural scenes. The nature of the covariance matrix of natural images was investigated by Field, who found that the spectrum of covariance matrix is proportional to the inverse of the square of the frequency.

We assume a circular hard boundary to the receptive fields, with a radius equal to the zero crossing of the correlation function.

We find that the solutions are the Fourier-Bessel functions. We will show in section 3, that under the assumption that the covariance matrix spectrum has a small non-rotationally symmetric correction, the solutions have a definite phase.

### 2.1 The Rotationally Symmetric Solution

The principal components are the eigen-functions of the covariance matrix. Therefore the equation we try to solve is the eigenvalue problem, i.e., the eigen-equation, which has the form

$$\sum_j C_{ij} W_j = \lambda W_i \quad (1)$$

where  $W_i$  are eigen-vectors,  $\lambda$  is the eigenvalue, and  $C_{ij}$  is the covariance matrix which is defined as  $C_{ij} = E[(I_i - E[I_i])(I_j - E[I_j])]$  for input pattern  $\{I_i\}$ . Since we are dealing with two dimensional space, the index  $i$  really denotes a point in the two dimensional space, so it is more convenient to rewrite the covariance matrix in the form  $C(\mathbf{r}_i, \mathbf{r}'_j)$ . Due to translational invariance,  $C(\mathbf{r}_i, \mathbf{r}'_j) = C(\mathbf{r}_i - \mathbf{r}'_j)$ . In the continuous limit, the summation will become an integral over  $\mathbf{r}'$ , thus the eigen-equation becomes

$$\int C(\mathbf{r} - \mathbf{r}') \psi(\mathbf{r}') d^2 \mathbf{r}' = \lambda \psi(\mathbf{r}). \quad (2)$$

in which  $w(\mathbf{r})$  is the continuous limit of the eigen-vectors  $W_i$ .

The Fourier transform (spectrum) of the covariance matrix has the form,  $C(\mathbf{k}) = c/\mathbf{k}^2$  where  $c$  is a constant. Hereafter we will set  $c = 1$  for convenience. Thus  $C(\mathbf{r})$  satisfies

$$\nabla^2 C(\mathbf{r} - \mathbf{r}') = -\delta(\mathbf{r} - \mathbf{r}'). \quad (3)$$

which can be readily proven by taking Fourier transformation on both side of this equation. Since the correlation function is zero on some boundary assumed to be a circular boundary

of radius  $a$ , then within this boundary it can be represented as a sum of a complete set of functions with the same boundary condition. We will choose the Bessel Fourier set  $W_{mi}$  which is zero on the boundary, which take the form

$$W_{mi}(\mathbf{r}) = \begin{cases} J_m(\frac{r}{\sqrt{\lambda_{mi}}}) \begin{cases} \cos(m\theta) \\ \sin(m\theta) \end{cases} & \text{for } r \leq a \\ 0 & \text{for } r > a \end{cases} \quad (4)$$

in which  $m = 0, 1, 2, \dots$ ,  $J_m(x)$  is the standard Bessel functions,  $\lambda_{mi}$  is the  $i$ th root of equation  $J_m(a/\sqrt{\lambda}) = 0$ ,  $r$  and  $\theta$  are the polar coordinates of  $\mathbf{r}$ . These functions solve the differential equation,

$$\nabla^2 W_{im} = -(1/\lambda_{mi}) W_{im}. \quad (5)$$

In this representation the correlation function must take the form

$$C(\mathbf{r} - \mathbf{r}') = \sum_{im} \lambda_{im} W_{im}^*(\mathbf{r}) W_{im}(\mathbf{r}') \quad (6)$$

Since, remembering that  $\delta(\mathbf{r} - \mathbf{r}') = \sum_{kl} W_{kl}^*(\mathbf{r}) W_{kl}(\mathbf{r}')$ , it is easy to see that  $C(\mathbf{r} - \mathbf{r}')$  is a solution of eq:3. It is important to notice that this solution to  $C(\mathbf{r} - \mathbf{r}')$  is not unique, since we can add a constant to this and still retain a radially symmetric equation. This is avoided by choosing the boundary  $a$  such that this constant is 0, which implies that the hard wired connections between retina and neurons must have a spatial extent which is equal to the zero crossing of the correlation function.

Thus plugging the correlation function of eq:6 into the eigen equation, representing the eigenfunctions as well as sums of this complete set,  $\psi(\mathbf{r}) = \sum_{jn} B_{jn} W_{jn}(\mathbf{r})$ , and using the orthogonality of these functions over the interval, we obtain that

$$\sum_{kl} \lambda_{kl} B_{kl} W_{kl}(\mathbf{r}) = \lambda \sum_{kl} B_{kl} W_{kl}(\mathbf{r}).$$

For which the solution, is that only one of the coefficients  $B_{ij} = 1$  and the rest are zero. The corresponding eigenvalue is  $\lambda = \lambda_{ij}$ . Thus the solutions are the Bessel, Fourier functions.

$$\begin{aligned} w_{mi}^1(\mathbf{r}) &= \begin{cases} J_m(\frac{r}{\sqrt{\lambda_{mi}}}) \cos(m\theta + \phi_{mi}) & \text{for } r \leq a \\ 0 & \text{for } r > a \end{cases} \\ w_{mi}^2(\mathbf{r}) &= \begin{cases} J_m(\frac{r}{\sqrt{\lambda_{mi}}}) \sin(m\theta + \phi_{mi}) & \text{for } r \leq a \\ 0 & \text{for } r > a \end{cases} \end{aligned} \quad (7)$$

where  $\phi_{mi}$  is a set of undetermined phases. These two eigen-functions have the same eigenvalue  $\lambda_{mi}$ , i.e., they are degenerate.

If we order the solutions by the magnitudes of the correspondent eigenvalues  $\lambda_{mi}$ , the first ten solutions,  $w_{mi}^1(\mathbf{r})$  with  $\phi_{mi} = 0$  and  $a = 1$ , are drawn in figure 8.

## 2.2 Retrieving the Phase

The solutions above  $w_{mi}^1(\mathbf{r})$  and  $w_{mi}^2(\mathbf{r})$  not only have undetermined phases, but also are degenerate. This contradicts the results of the simulations performed by Hancock in which the phases seem to always take the value zero, and the  $W_{mi}^1$  solution has a different eigenvalue from the  $W_{mi}^2$  solution. These results can be retrieved if we assume that the covariance matrix has a non-rotationally symmetric perturbation term. This assumption is not arbitrary since an inspection of Fields results reveals that this is indeed the case. Hereafter we assume this perturbation term has, in  $\mathbf{k}$  space, the form

$$C'(\mathbf{k}) = U(k)T(\theta_{\mathbf{k}}). \quad (8)$$

In order to calculate this perturbation, the representation of this perturbation in the two degenerate eigen-functions  $W_{mi}^1(\mathbf{r})$  and  $W_{mi}^2(\mathbf{r})$  has to be calculated. It is easier to perform this in  $\mathbf{k}$  space in which the eigen-functions  $W_{mi}^1(\mathbf{r})$  and  $W_{mi}^2(\mathbf{r})$  are replaced by their Fourier transforms,

$$\begin{aligned} W_{mi}^1(\mathbf{k}) &= f_{mi}(k)\cos(m\theta_{\mathbf{k}} + \phi_{mi}) \\ W_{mi}^2(\mathbf{k}) &= f_{mi}(k)\sin(m\theta_{\mathbf{k}} + \phi_{mi}) \end{aligned} \quad (9)$$

in which

$$f_{mi}(k) = \pi j^m \int_0^a J_m\left(\frac{r}{\sqrt{\lambda_{mi}}}\right) J_m(kr) r dr \quad (10)$$

where  $j^2 = -1$ . If we denote

$$T(\theta_{\mathbf{k}}) = \sum_l t_l \cos(l(\theta_{\mathbf{k}} - \alpha_l)) \quad (11)$$

which is the Fourier expansion of  $T(\theta_{\mathbf{k}})$ . The representation of the perturbation matrix with respect to the two degenerate eigen-functions has the form

$$\begin{aligned} (C'_{(\mu, m, i | \gamma, m, i)})_{(\mu=1,2 | \gamma=1,2)} &= \left( \int W_{mi}^\mu(\mathbf{k})^* C'(\mathbf{k}) W_{mi}^\gamma(\mathbf{k}) d^2\mathbf{k} \right)_{(\mu=1,2 | \gamma=1,2)} \\ &= g_{mi} \begin{pmatrix} \cos(\delta) & \sin(\delta) \\ \sin(\delta) & -\cos(\delta) \end{pmatrix} \end{aligned} \quad (12)$$

in which  $\delta = 2\phi_{mi} + 2m\alpha_{2m}$  and  $g_{mi} = \frac{\pi}{2} t_{2m} \int U(k) |f_{mi}(k)|^2 k dk$ . Since the two eigen-functions are degenerate, any linear combination of these two eigen-functions is an eigen-function of  $C$ . Therefore, all we have to do is to find a linear combination of them which diagonalizes the perturbation matrix, i.e., to find the eigenvalues and eigen-vectors of the matrix in equation 12, which are

$$\begin{aligned} &\begin{pmatrix} \cos(\delta/2) \\ \sin(\delta/2) \end{pmatrix} \\ &\begin{pmatrix} -\sin(\delta/2) \\ \cos(\delta/2) \end{pmatrix} \end{aligned} \quad (13)$$

with eigenvalues  $g_{mi}$  and  $-g_{mi}$  respectively. Furthermore, if  $U(k) = \epsilon/k^2$  then the complete expression for the correction to the eigenvalue takes the form  $g_{mi} = \epsilon\lambda_{mi}t_{2m}/2$ .

Thus the eigen-functions and eigenvalues after the perturbation can be readily written out as

$$\begin{aligned} W_{mi}^+(\mathbf{k}) &= J_m\left(\frac{r}{\sqrt{\lambda_{mi}}}\right)\cos(m(\theta - \alpha_{2m})) \\ W_{mi}^-(\mathbf{k}) &= J_m\left(\frac{r}{\sqrt{\lambda_{mi}}}\right)\sin(m(\theta - \alpha_{2m})) \end{aligned} \tag{14}$$

with eigenvalues  $\lambda_{mi}^+ = \lambda_{mi} + g_{mi}$ , and  $\lambda_{mi}^- = \lambda_{mi} - g_{mi}$ , respectively. So the degeneracy is broken. This is in agreement with Hancock's simulations. These solutions have an important feature, i.e., their phases are determined by the properties of the real world covariance matrix. If the covariance matrix has a definite symmetry with an inclination angle  $\alpha$ , then the solutions would also have the same symmetry angle. Because in this case  $\alpha_{2m} = \alpha$  for all  $m$ . The spectrums of the covariance matrix, shown in figure 7 of Field's paper, indeed indicates a symmetry axis along  $\alpha = 0$ . Thus equation 14 predicts the zero phase result found in Hancock's simulation. When Hancock used images which were tilted by 45 degrees before being scanned, the preferred axis of the receptive fields was found to be 45 degrees. Again this is predicted by equation 14, because the symmetry axis of the covariance matrix spectrum also gets rotated by 45 degrees due to the rotated images, i.e.,  $\alpha = 45^\circ$ , and thus the solutions also get rotated by 45 degrees.

### 2.3 Discussion

We have calculated the forms of the principal components of natural images based on the result about the covariance matrix, and have shown that a non-rotationally symmetric perturbation can break the degeneracy and give a definite phase which only depends on the properties of the real world covariance matrix. These results for a large part agree with the numerical simulation.

The neurobiological relevance of the type of technique used in this paper is that we can deduce for different learning rules what kinds of receptive fields they should produce. Given these receptive fields, we can compare them to the real biological receptive fields. This comparison can be used to assess whether the biological hardware really implements or approximates a theoretically proposed learning rule.

The most obvious conclusion which stands out when we observe the results in figure 8, is that these receptive fields have little resemblance to receptive fields reported in the biological literature. Does this imply that biological neurons are not principal component analyzers? When addressing this question we have to keep in mind that the natural images projected on the retina, undergo preprocessing in the retina and LGN, before they reach the visual cortex. Similar preprocessing should therefore be applied to natural images in simulations and analytic studies, before a sensible answer can be given.

### 3 Hybrid Network Techniques

We have previously shown that hybrid network techniques can significantly improve network performance on difficult real-world problems. Below, we develop a firm mathematical framework for the observed network performance improvement.

#### 3.1 Basic Ensemble Method

Consider the following regression problem

$$y = f(x) + n$$

where  $y$  is a random variable with mean  $f(x) = E[y|x]$  and  $n$  is independent zero-mean noise.<sup>1</sup> We present the Basic Ensemble Method (BEM) which combines a population of regression estimates,  $\hat{f}_i(x)$ , to estimate a function  $f(x)$ .

Suppose that we have two finite data sets whose elements are all independent and identically distributed random variables: a training data set  $\mathcal{A} = \{(x_m, y_m)\}$  and a cross-validatory data set  $\mathcal{CV} = \{(x_l, y_l)\}$ . Further suppose that we have used  $\mathcal{A}$  to generate a set of functions,  $\mathcal{F} = \{f_i(x)\}$ , each element of which approximates  $f(x)$ .<sup>2</sup> We would like to find the best approximation to  $f(x)$  using  $\mathcal{F}$ .

One common choice is to use the *naive estimator*,  $f_{\text{Naive}}(x)$ , which minimizes the empirical mean square error relative to  $f(x)$ ,<sup>3</sup>

$$\text{MSE}[f_i] = E_{\text{CV}}[(y_l - f_i(x_l))^2],$$

thus

$$f_{\text{Naive}}(x) = \arg \min_i \{\text{MSE}[f_i]\}.$$

This choice is unsatisfactory for two reasons: First, in selecting only one regression estimate from the population of regression estimates represented by  $\mathcal{F}$ , we are discarding potentially useful information that is stored in the discarded regression estimates; second, since the  $\mathcal{CV}$  data set is random, there is a certain probability that some other network from the population will perform better than the naive estimate on some other previously unseen data set sampled from the same distribution. A more reliable estimate of the performance on previously unseen data is the average of the performances over the population  $\mathcal{F}$ . Below, we will see how we can avoid both of these problems by using the BEM estimator,  $f_{\text{BEM}}(x)$ , and thereby generate an improved regression estimate.

---

<sup>1</sup>The noise for minimizing the MSE is assumed to be Gaussian; but this assumption is not necessary for what follows.

<sup>2</sup>For our purposes, it does not matter how  $\mathcal{F}$  was generated, unlike Monte Carlo. In practice we will use a set of backpropagation networks trained on the  $\mathcal{A}$  data set but started with different random weight configurations. This replication procedure is standard practice when trying to optimize neural networks.

<sup>3</sup>Here, and in all of that follows, the expected value is taken over the cross-validatory set  $\mathcal{CV}$ .



Define the *misfit* of function  $f_i(x)$ , the deviation from the true solution, as  $m_i(x) \equiv f(x) - f_i(x)$ . The empirical mean square error can now be written in terms of  $m_i(x)$  as

$$\text{MSE}[f_i] = E[m_i^2].$$

The average mean square error is therefore

$$\overline{\text{MSE}} = \frac{1}{N} \sum_{i=1}^N E[m_i^2].$$

Define the BEM regression function,  $f_{\text{BEM}}(x)$ , as

$$f_{\text{BEM}}(x) \equiv \frac{1}{N} \sum_{i=1}^N f_i(x) = f(x) - \frac{1}{N} \sum_{i=1}^N m_i(x)$$

If we now assume that the  $m_i(x)$  are mutually independent with zero mean, we can calculate the mean square error of  $f_{\text{BEM}}(x)$  as

$$\begin{aligned} \text{MSE}[f_{\text{BEM}}] &= E\left[\left(\frac{1}{N} \sum_{i=1}^N m_i\right)^2\right] \\ &= \frac{1}{N^2} E\left[\sum_{i=1}^N m_i^2\right] + \frac{1}{N^2} E\left[\sum_{i \neq j} m_i m_j\right] \\ &= \frac{1}{N^2} E\left[\sum_{i=1}^N m_i^2\right] + \frac{1}{N^2} \sum_{i \neq j} E[m_i] E[m_j] \\ &= \frac{1}{N^2} E\left[\sum_{i=1}^N m_i^2\right], \end{aligned} \tag{15}$$

which implies that

$$\text{MSE}[f_{\text{BEM}}] = \frac{1}{N} \overline{\text{MSE}}. \tag{16}$$

This is a powerful result because it tells us that by averaging regression estimates, we can reduce our mean square error by a factor of  $N$  when compared to the population performance: By increasing the population size, we can in principle make the estimation error arbitrarily small! In practice however, as  $N$  gets large our assumptions on the misfits,  $m_i(x)$ , eventually breakdown. In particular, the assumption that  $E[m_i m_j] = E[m_i] E[m_j]$  is no longer valid.

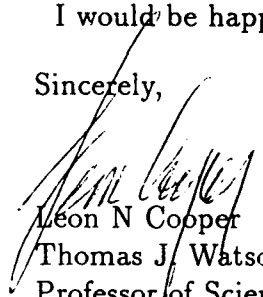
Consider the individual elements of the population  $\mathcal{F}$ . These estimators will more or less follow the true regression function. If we think of the misfits functions as random noise functions added to the true regression function and these noise functions are uncorrelated with zero mean, then the averaging of the individual estimates is like averaging over the noise. In this sense, the ensemble method is smoothing in functional space and can be thought of as a regularizer with a smoothness assumption on the true regression function.

An additional benefit of the ensemble method's ability to combine multiple regression estimates is that the regression estimates can come from many different sources. This fact

allows for flexibility in the application of the ensemble method. For example, the regression estimates can have different functional forms; or can be selected using different optimization (i.e. "training") algorithms; or can be selected by optimizing over different data sets. This last option - optimizing on different data sets - has important ramifications. One standard method for avoiding over-fitting during training is to use a cross-validators hold-out set. The cross-validators hold-out set is a subset of the total data available to us and is used to determine when to stop training. The hold-out data is not used to train. The problem is that since we use cross-validation to avoid over-fitting, each regression estimate is never trained on the hold-out data (i.e. the cross-validators data set) and therefore, each regression estimate "sees" only part of the data and may be missing valuable information about the distribution of the data particularly if the total data set is small. This will always be the case for a single regression estimate using a cross-validators stopping rule. However, this is not a problem for the ensemble estimator. When constructing our population,  $\mathcal{F}$ , we can train each regression estimate on the entire training set and let the smoothing property of the ensemble process remove any over-fitting or we can train each regression estimate in the population with a different split of training and hold-out data. In this way, the population as a whole will have seen the entire data set while each regression estimate has avoided over-fitting by using a cross-validators stopping rule. Thus the ensemble estimator will see the entire data set while the naive estimator will not. In general, with this framework we can now easily extend the statistical jackknife, bootstrap and cross-validation techniques (Efron, 1982; Miller, 1974; Stone, 1974) to find better regression functions.

I would be happy to answer any questions you might have concerning this report.

Sincerely,



Leon N Cooper

Thomas J. Watson, Sr.

Professor of Science

Director, Institute for Brain and Neural Science

Enclosure: Publication List

## References

- Efron, B. (1982). *The Jackknife, the Bootstrap and Other Resampling Plans*. SIAM, Philadelphia, PA.
- Jones, J. P. and Palmer, L. A. (1987). The two-dimensional spatial structure of simple receptive fields in cat striate cortex. *Journal of Neurophysiology*, 58(6):1187-1258.
- Miller, R. G. (1974). The jackknife - a review. *Biometrika*, 61(1):1-16.
- Stone, M. (1974). Cross-validatory choice and assessment of statistical predictions (with discussion). *J. Roy. Stat. Soc. Ser. B*, 36:111-147.

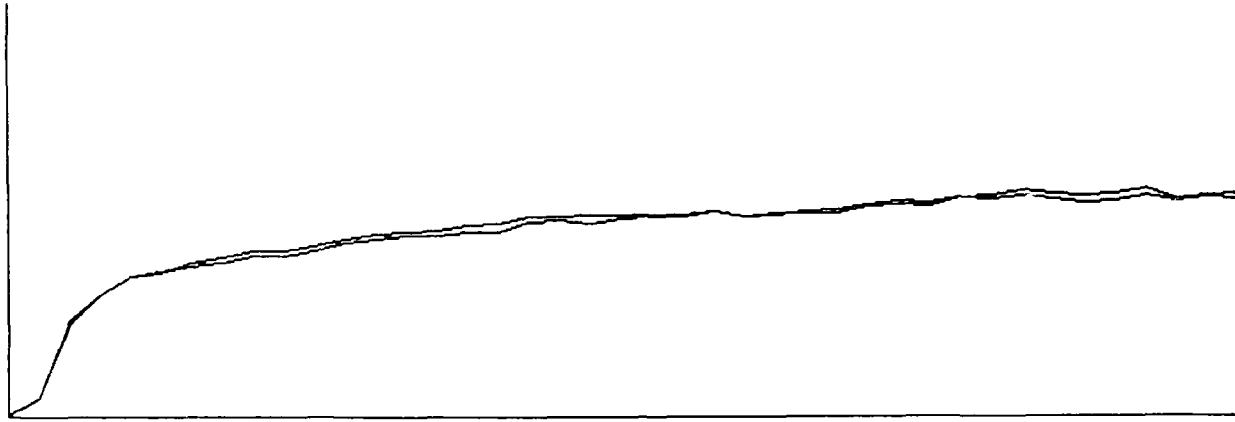


Figure 2: CBC simulation of normal rearing. The graph displays the maximum response to the training data for the left and right eyes.

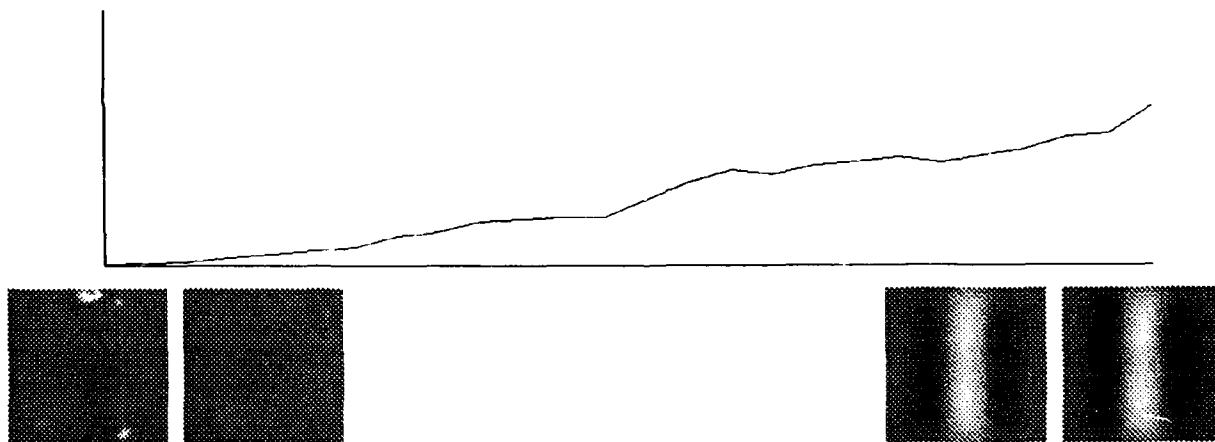


Figure 3: Simulation of normal rearing with realistic input. The 2d maps are the receptive fields for the left and right eyes at the beginning and end of the simulation. The upper graph shows the maximum of the left and right eye maps throughout the simulation.

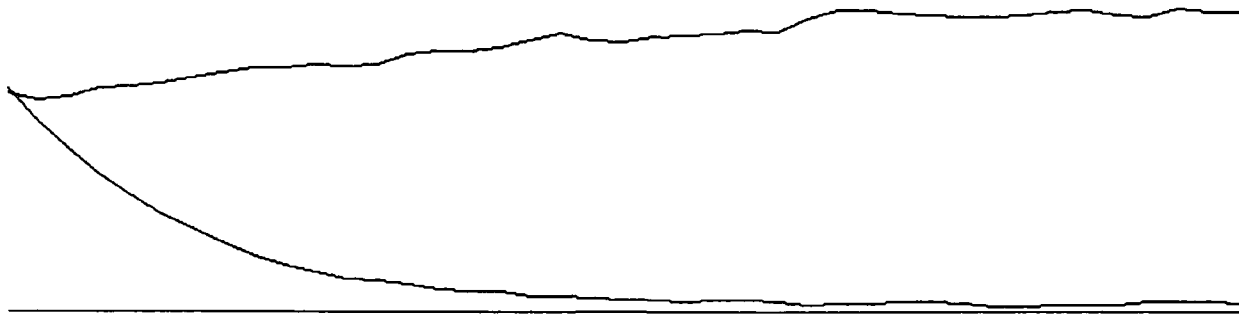


Figure 4: CBC simulation of monocular deprivation. The graph displays the maximum response to the training data for the left and right eyes.

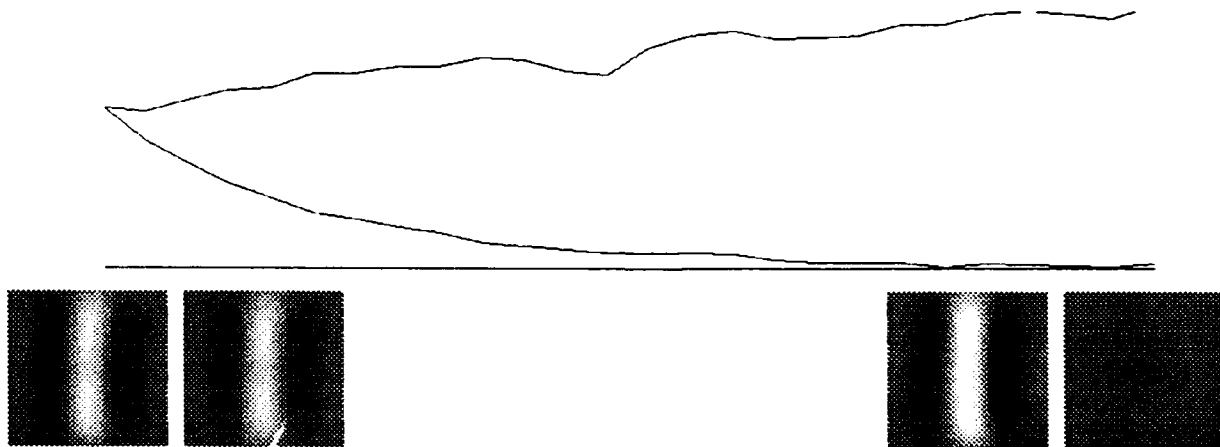


Figure 5: Simulation of monocular deprivation with realistic input. The 2d maps are the receptive fields for the left and right eyes at the beginning and end of the simulation. The upper graph shows the maximum of the left and right eye maps throughout the simulation.



Figure 6: CBC simulation of reverse suture. The graph displays the maximum response to the training data for the left and right eyes.

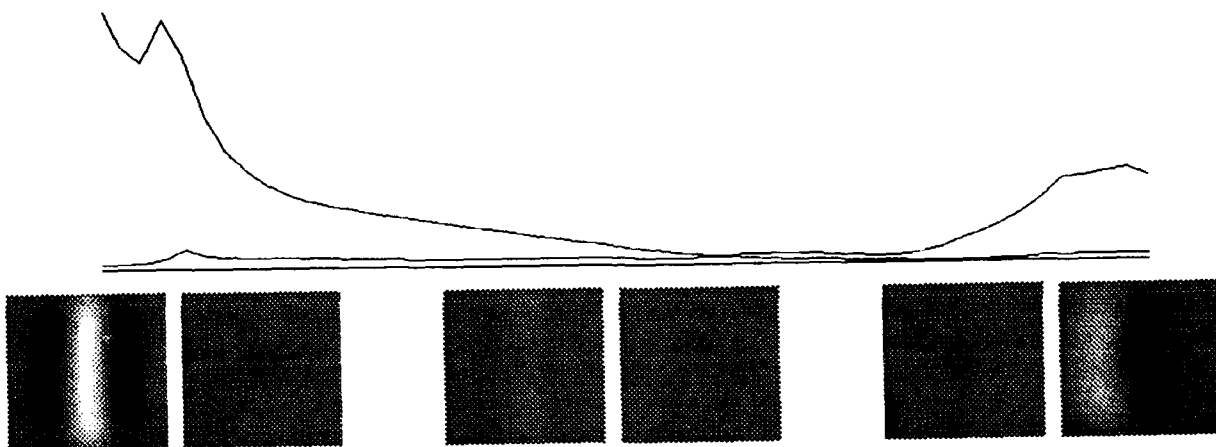


Figure 7: Simulation of reverse suture with realistic input. The 2d maps are the receptive fields for the left and right eyes at the beginning, middle and end of the simulation. The upper graph shows the maximum of the left and right eye maps throughout the simulation.

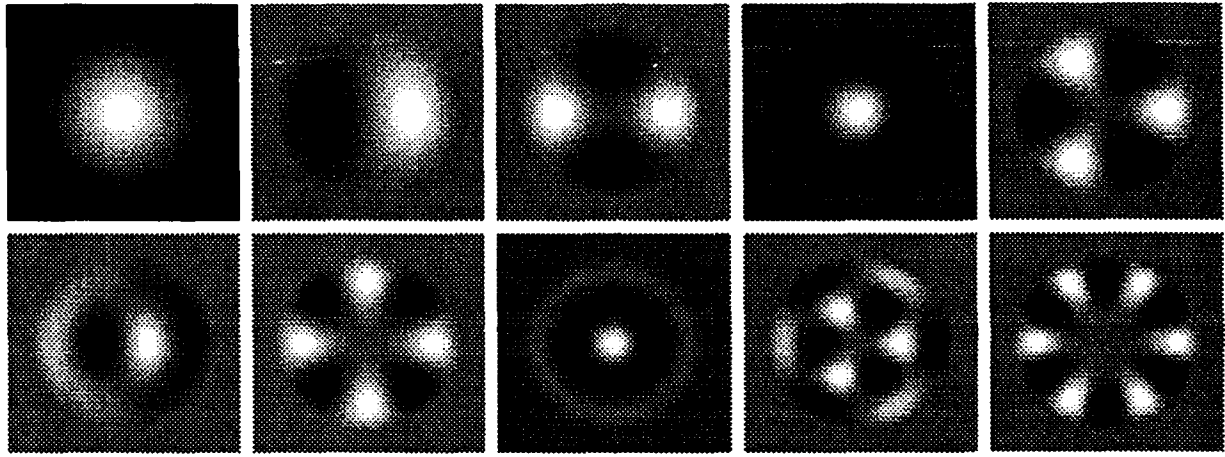


Figure 8: The shapes of the first ten principal components. ( $W_{mi}^1(\mathbf{r})$  with  $a = 1$  and  $\phi_{mi} = 0$ )

## SOME ONR SUPPORTED PUBLICATIONS

- [1] E. Bienenstock, L. N Cooper, and P. Munro. On the development of neuron selectivity: Orientation specificity and binocular interaction in visual cortex. *Journal of Neuroscience*, 2:32-48, 1982.
- [2] J. A. Anderson. Cognitive and psychological computation with neural models. *IEEE Transactions on Systems, Man, and Cybernetics*, 13(5):799-815, 1983.
- [3] M. F. Bear and J. D. Daniels. The plastic response to monocular deprivation persists in kitten visual cortex after chronic depletion of norepinephrine. *Journal of Neuroscience*, 3:407-416, 1983.
- [4] M. F. Bear, M. A. Paradiso, M. Schwartz, S. B. Nelson, K. M. Carnes, and J. D. Daniels. Two methods of catecholamine depletion in kitten visual cortex yield different effects on plasticity. *Nature*, 302:245-247, 1983.
- [5] M. A. Paradiso, M. F. Bear, and J. D. Daniels. Effects of intracortical infusion of 6-hydroxydopamine on the response of kitten visual cortex to monocular deprivation. *Experimental Brain Research*, 51:413-422, 1983.
- [6] J. A. Anderson. Neural models and a little about language. In D. Caplan, A. Roche-Lecourgs, and A. Smith, editors, *Biological Bases of Language*, pages 361-398. MIT Press, Cambridge, MA, 1984.
- [7] L. N Cooper. Neuron learning to network organization. In M. S. Berger, editor, *J. C. Maxwell Sesquicentennial Symposium*. Elsevier Science Publications, New York, 1984.
- [8] J. D. Daniels, E. Pressman, M. Schwartz, S. B. Nelson, and D. Kraus. Effects of luminance and flicker on ocular dominance shift in kitten visual cortex. *Exp. Brain Research*, 54:186-190, 1984.
- [9] A. Haycock and M. F. Bear. The stability of 6-hydroxydopamine under minipump conditions. *Experimental Brain Research*, 56:183-186, 1984.
- [10] A. G. Knapp and J. A. Anderson. A theory of categorization based on distributed memory storage. *Journal of Experimental Psychology, Learning, Memory, and Cognition*, 10(4):616-637, 1984.
- [11] P. W. Munro. A model for generalization and specification by single neurons. *Biological Cybernetics*, 51:169-179, 1984.
- [12] J. A. Anderson. What hebb synapses build. In W. Levy, S. Lehmkuhle, and J. Anderson, editors, *Synaptic Modification, Neuron Selectivity, and Nervous System Organization*. Lawrence Erlbaum Associates, Hillsdale, NJ, 1985.



- [13] M. F. Bear, K. M. Carnes, and F. F. Ebner. An investigation of cholinergic circuitry in cat striate cortex using acetylcholinesterase histochemistry. *Journal of Comparative Neurology*, 234:411-430, 1985.
- [14] M. F. Bear, K. M. Carnes, and F. F. Ebner. Postnatal changes in the distribution of acetylcholinesterase in kitten striate cortex. *Journal of Comparative Neurology*, 237:519-532, 1985.
- [15] M. F. Bear, D. E. Schmechel, and F. F. Ebner. Glutamic decarboxylase in the striate cortex of normal and monocularly deprived kittens. *Journal of Neuroscience*, 5:1262-1275, 1985.
- [16] L. N Cooper. A model for the development of neurons in visual cortex. In D. Rose and V. Dobson, editors, *Models of the Visual Cortex*. John Wiley and Sons, Ltd., New York, 1985.
- [17] L. N Cooper, P. W. Munro, and C. L. Scofield. Neuron selectivity: Single neuron and neuron networks. In W. Levy, S. Lehmkuhle, and J. Anderson, editors, *Synaptic Modification, Neuron Selectivity, and Nervous System Organization*. Lawrence Erlbaum Associates, Hillsdale, NJ, 1985.
- [18] C. F. Hohmann, M. F. Bear, and F. F. Ebner. Glutamic acid decarboxylase activity decreases in mouse neocortex after lesions of the basal forebrain. *Exp. Brain Research*, 333:165-168, 1985.
- [19] A. H. Kawamoto and J. A. Anderson. A neural network of multistable perception. *Acta Psychologica*, 59:35-65, 1985.
- [20] S. B. Nelson, M. A. Schwartz, and J. D. Daniels. Clonidine and cortical plasticity: New evidence for noradrenergic involvement. *Developmental Brain Research*, 23:39-50, 1985.
- [21] C. L. Scofield and L. N Cooper. Development and properties of neural networks. *Contemporary Physics*, 26(2):125-145, 1985.
- [22] J. A. Anderson. Cognitive capabilities of a parallel system. In E. Bienenstock, F. Fogelman-Soulie, and G. Weisbuch, editors, *Disordered Systems in Biological Organization*. Springer-Verlag, New York, 1986.
- [23] J. A. Anderson and G. Murphy. Psychological concepts in a parallel system. *Physica D*, 22:318-336, 1986.
- [24] L. N Cooper. Neuron learning to brain organization. *Cell Biophysics*, 9:103-144, 1986.
- [25] L. N Cooper. Theory of an immune system retrovirus. *Proceedings of the National Academy of Sciences*, 83:9159-9163, 1986.
- [26] A. Saul and J. D. Daniels. Modeling and simulation I: Introduction and guidelines. *Journal of Electrophysiological Techniques*, 13:95-109, 1986.
- [27] A. Saul and J. D. Daniels. Modeling and simulation II: Specificity models for visual cortex development. *Journal of Electrophysiological Techniques*, 13:211-231, 1986.

- [28] A. B. Saul and E. E. Clothiaux. Modeling and simulation iii: Simulation of a model for development of visual cortical specificity. *Journal of Electrophysiological Techniques*, 13:279-306, 1986.
- [29] C. M. Bachmann, L. N Cooper, A. Dembo, and O. Zeitouni. A relaxation model for memory with high storage density. *Proceedings of the National Academy of Sciences*, 84:7529-7531, 1987.
- [30] M. F. Bear, L. N Cooper, and F. F. Ebner. The physiological basis of a theory for synapse modification. *Science*, 237:42-48, 1987.
- [31] L. N Cooper. Cortical plasticity: Theoretical analysis, experimental results. In J. Rauschecker and P. Marler, editors, *Imprinting and Cortical Plasticity*. John Wiley and Sons, Ltd., New York, 1987.
- [32] L. N Cooper. Future of brain and information research. *Naval Research Reviews*, 1987.
- [33] L. N Cooper. Memory, distributed. In G. Edelman, editor, *Encyclopedia of Neuroscience*, volume 2, pages 633-634. Birkhauser, Boston, MA, 1987.
- [34] J. P. Donoghue and J. N. Sanes. Peripheral nerve injury in developing rats reorganizes motor cortex. *Proceedings of the National Academy of Sciences*, 84:1123-1126, 1987.
- [35] A. Kleinschmidt, M. F. Bear, and W. Singer. Blockade of NMDA receptors disrupts experience-dependent plasticity of kitten striate cortex. *Science*, 238:355-358, 1987.
- [36] M. F. Bear. Involvement of excitatory amino acid receptor mechanisms in the experience-dependent development of visual cortex. In E. Cavalheiro, J. Lehamn, and L. Turski, editors, *Frontiers in Excitatory Amino Acid Research*, pages 393-401. Liss, New York, 1988.
- [37] M. F. Bear, A. Kleinschmidt, and W. Singer. Experience-dependent modifications of kitten striate cortex are not prevented by thalamic lesions that include the intralaminar nuclei. *Experimental Brain Research*, 70:627-631, 1988.
- [38] L. N Cooper. Disability unbound: Challenge to the brain and information science and technologies. In D. Cone and D. Galamaga, editors, *Proceedings of a Symposium held November 4, 1987 at the Rhode Island Medical Center in Cranston, Rhode Island, Donna M. Cone, Ph. D. and Donald P. Galamaga, eds. (Copyright by the Department of Mental Health, Retardation, and Hospitals. Rhode Island Department of Mental Health, Retardation and Hospitals, Cranston, RI, 1988.*
- [39] L. N Cooper. Local and global factors in learning. In J. Davis, R. Newburgh, and E. Wegman, editors, *Brain Structure, Learning and Memory*, pages 171-191. Westview Press, Boulder, CO, 1988.
- [40] L. N Cooper. Some recent developments in the theory of neural networks. In R. Cotterill, editor, *Computer Simulations in Brain Science*, pages 1-11. Cambridge University Press, New York, 1988.

- [41] L. N Cooper and C. L. Scofield. Mean field theory of a neural network. *Proceedings of the National Academy of Sciences*, 85:1973-1977, 1988.
- [42] A. Dembo and O. Zeitouni. General potential surfaces and neural networks. *Physical Review A*, 37:2134-2143, 1988.
- [43] N. Intrator, G. P. DeoCampo, and L. N. Cooper. Analysis of immune system retrovirus equations. In A. S. Perleson, editor, *Theoretical Immunology, Part two, SFI Studies in the Sciences of Complexity*. Addison-Wesley Publishing Co, 1988.
- [44] J. N. Sanes, S. Suner, J. F. Lando, and J. Donoghue. Rapid reorganization of adult rat motor cortex somatic representation patterns after motor nerve injury. *Proceedings of the National Academy of Sciences*, 85:2003-2007, 1988.
- [45] M. F. Bear, L. N Cooper, and F. F. Ebner. A physiological basis for synaptic modification. In K. Kelner and D. Koshland, Jr., editors, *Molecules to Models: Advances in Neuroscience*. American Association for the Advancement of Science, Washington, 1989.
- [46] L. N Cooper. First word. *Omni Magazine*, 11(6), 1989.
- [47] L. N Cooper, M. F. Bear, and F. F. Ebner. Synaptic modification model of learning and memory. In G. Adelman, editor, *Neuroscience Year, Supplement 1 to the Encyclopedia of Neuroscience*, pages 156-160. Birkhauser, Boston, MA, 1989.
- [48] J. P. Donoghue and J. N. Sanes. Organization of adult motor cortex representation patterns following neonatal forelimb nerve injury in rats. *Journal of Neuroscience*, 8:3221-3233, 1989.
- [49] S. M. Dudek and M. F. Bear. A biochemical correlate of the critical period for synaptic modification in the visual cortex. *Science*, 246:673-675, 1989.
- [50] S. M. Dudek, W. D. Bowen, and M. F. Bear. Postnatal changes in glutamate stimulated phosphoinositide turnover in rat neocortical synaptoneurosomes. *Developmental Brain Research*, 47:123-128, 1989.
- [51] Q. Gu, M. F. Bear, and W. Singer. Blockade of NMDA-receptors prevents ocularity changes in kitten visual cortex after reversed monocular deprivation. *Developmental Brain Research*, 47:281-288, 1989.
- [52] R. L. Neve and M. F. Bear. Visual experience regulates gene expression in the developing visual cortex. *Proceedings of the National Academy of Sciences*, 86:4781-4784, 1989.
- [53] K. E. Barsted and M. F. Bear. Basal forebrain projections to somatic sensory cortex in the cat. *Journal of Neurophysiology*, 64:1223-1232, 1990.
- [54] M. F. Bear and H. Colman. Binocular competition in the control of geniculate cell size depends upon visual cortical NMDA receptor activation. *Proceedings of the National Academy of Sciences*, 87:9246-9249, 1990.
- [55] M. F. Bear and L. N Cooper. Molecular mechanisms for synaptic modification in the visual cortex: Interaction between theory and experiment. In M. Gluck and D. Rumelhart, editors, *Neuroscience and Connectionist Theory*. Lawrence Erlbaum Associates, Hillsdale, NJ, 1990.

- [56] M. F. Bear, A. Kleinschmidt, Q. Gu, and W. Singer. Disruption of experience-dependent synaptic modification in the striate cortex by infusion of an NMDA receptor antagonist. *Journal of Neuroscience*, 10(3):909-925, 1990.
- [57] L. N Cooper, M. Bear, F. Ebner, and C Scofield. Neural networks: from theorems to test tubes. In J. McGaugh, N. Weinberger, and G. Lynch, editors, *Brain Organization and Memory: cells, systems and circuits*. Oxford University Press, New York, 1990.
- [58] L. N Cooper and D. L. Reilly. An overview of neural networks: Early models to real world systems. In S. Zournetzer, J. Davis, and C. Lau, editors, *An Introduction to Neural and Electronic Networks*. Academic Press, San Diego, 1990.
- [59] R. W. Dikes, N. Tremblay, and M. F. Bear. Cholinergic modulation of synaptic plasticity in sensory neocortex. In R. Richardson, editor, *Activation to Aquisition: Functional Aspects of the Basal Forebrain Cholinergic System*. Birkhauser, Boston, MA, 1990.
- [60] D. Feldman, J. E. Sherin, W. Press, and M. F. Bear. NMDA stimulated calcium uptake by kitten visual cortex *in vitro*. *Experimental Brain Research*, 80:252-259, 1990.
- [61] N. Intrator. Feature extraction using an unsupervised neural network. In D. S. Touretzky, J. L. Ellman, T. J. Sejnowski, and G. E. Hinton, editors, *Proceedings of the 1990 Connectionist Models Summer School*, pages 310-318. Morgan Kaufmann, San Mateo, CA, 1990.
- [62] N. Intrator. A neural network for feature extraction. In D. S. Touretzky and R. P. Lippmann, editors, *Advances in Neural Information Processing Systems*, volume 2, pages 719-726. Morgan Kaufmann, San Mateo, CA, 1990.
- [63] S. L. Juliano, M. F. Bear, W. Ma, and D. Elsin. Cholinergic manipulation alters stimulus-evoked metabolic activity in cat somatosensory cortex. *Journal of Computational Neurology*, 297:106-120, 1990.
- [64] M. F. Bear. Involvement of excitatory amino acid receptor mechanisms in visual cortical plasticity. In B. Meldrum, F. Moroni, R. Simon, and J Woods, editors, *Fidia Research Foundation Symposium Series*, volume 5. Raven Press, 1991.
- [65] M. F. Bear. Recent progress toward an understanding of experience-dependent visual cortical plasticity at the molecular level. In A. Gorea, editor, *Representations of Vision: Trends and Tacit Assumptions in Vision Research*. Cambridge University Press, 1991.
- [66] M. F. Bear. Use of developing visual cortex as a model to study the mechanisms of experience-dependent synaptic plasticity. *Brain Research Reviews*, 16:198-200, 1991.
- [67] M. F. Bear and S. Dudek. Stimulation of phosphoinositide turnover by excitatory amino acids: pharmacology, development and role in visual cortical plasticity. In J. Wolpaw, J. Schmidt, and T. Vaughan, editors, *Activity-driven CNS changes in learning and development - Ann. N. Y. Acad. Sci.*, volume 627. N. Y. Acad. Sci., 1991.
- [68] M. F. Bear and S. M. Dudek. Excitatory amino acid stimulated phosphoinositide turnover: Pharmacology, development and role in visual cortical plasticity. *Annals of the New York Academy of Science*, 627:42-56, 1991.

- [69] E. E. Clothiaux, L. N Cooper, and M. F. Bear. Synaptic plasticity in visual cortex: Comparison of theory with experiment. *Journal of Physiology*, 66:1785-1804, 1991.
- [70] L. N Cooper. Brain and information research. In *Encyclopedia of Computer Science Technology: Supplement 8*, volume 23, pages 35-47. Marcel Dekker, Inc., New York, 1991.
- [71] L. N Cooper. Foreword. In P. Antognette and V. Milutinovic, editors, *Neural Networks: Concepts, Applications and Implementations*. Prentice-Hall, New Jersey, 1991.
- [72] L. N Cooper. Hybrid neural network architectures: Equilibrium systems the pay attention. In R. J. Mammone and Y. Zeevi, editors, *Neural Networks and Applications*, pages 81-96. Academic Press, San Diego, 1991.
- [73] L. N Cooper. Scientific fraud. *George Street Journal*, 16(15), 1991.
- [74] L. N Cooper. Visual cortex: Window on the biological basis of learning and memory. In H. Wechsler, editor, *Neural Networks for Human and Machine Perception*. Academic Press, San Diego, 1991.
- [75] N. Intrator. Exploratory feature extraction in speech signals. In R. P. Lippmann, J. E. Moody, and D. S. Touretzky, editors, *Advances in Neural Information Processing Systems*, volume 3, pages 241-247. Morgan Kaufmann, San Mateo, CA, 1991.
- [76] N. Intrator. Localized exploratory projection pursuit. In Ed Wegman, editor, *Computer Science and Statistics: Proceedings of the 23rd Symposium on the Interface*, pages 237-240. Amer. Statist. Assoc., Washington, DC., 1991.
- [77] N. Intrator, J. I. Gold, H. H. Bülthoff, and S. Edelman. Three-dimensional object recognition using an unsupervised neural network: Understanding the distinguishing features. In Y. Feldman and A. Bruckstein, editors, *Proceedings of the 8th Israeli Conference on AICV*, pages 113-123. Elsevier, 1991.
- [78] N. Intrator and G. Tajchman. Supervised and unsupervised feature extraction from a cochlear model for speech recognition. In B. H. Juang, S. Y. Kung, and C. A. Kamm, editors, *Neural Networks for Signal Processing - Proceedings of the 1991 IEEE Workshop*, pages 460-469. IEEE Press, New York, NY, 1991.
- [79] M. P. Perrone. A novel recursive partitioning criterion. In *Proceedings of the International Joint Conference on Neural Networks*, volume II, page 989. IEEE, 1991.
- [80] I. J. Reynolds and M. F. Bear. The effects of age and visual experience on [3H] MK801 bindings to NMDA receptors in the kitten visual cortex. *Experimental Brain Research*, 85:611-615, 1991.
- [81] M. F. Bear, W. A. Press, and B. W. Connors. Long-term potentiation of slices of kitten visual cortex and the effects of NMDA receptor blockade. *Journal of Neurophysiology*, 67:841-851, 1992.
- [82] S. M. Dudek and M. F. Bear. Homosynaptic long-term depression in area CA1 of hippocampus and the effects on NMDA receptor blockade. *Proc. Natl. Acad. Sci.*, 89:4363-4367, 1992.

- [83] N. Intrator. Feature extraction using an unsupervised neural network. *Neural Computation*, 4:98-107, 1992.
- [84] N. Intrator and L. N. Cooper. Objective function formulation of the BCM theory of visual cortical plasticity: Statistical connections, stability conditions. *Neural Networks*, 5:3-17, 1992.
- [85] N. Intrator, J. I. Gold, H. H. Bülthoff, and S. Edelman. 3D Object recognition using unsupervised feature extraction. In J. E. Moody, S. J. Hanson, and R. P. Lippmann, editors, *Advances in Neural Information Processing Systems*, volume 4, pages 460-467. Morgan Kaufmann, San Mateo, CA, 1992.
- [86] M. P. Perrone. A soft-competitive splitting rule for adaptive tree-structured neural networks. In *Proceedings of the International Joint Conference on Neural Networks*, volume IV, pages 689-693. IEEE, 1992.
- [87] M. P. Perrone and N. Intrator. Unsupervised splitting rules for neural tree classifiers. In *Proceedings of the International Joint Conference on Neural Networks*, volume III, pages 820-825. IEEE, 1992.
- [88] G. N. Tajchman and N. Intrator. Phonetic classification of TIMIT segments preprocessed with Lyon's cochlear model using a supervised/unsupervised hybrid neural network. In *Proceedings International Conference on Spoken Language Processing*, Banff, Alberta, Canada, October 1992.
- [89] J. M. Gilchrist, M. P. Perrone, and J. Ross. Is neuromuscular transmission jitter a non-linear dynamical process? In *Fifth International Conference on Computerized and Quantitative EMG*, 1993.
- [90] N. Intrator. Combining exploratory projection pursuit and projection pursuit regression with application to neural networks. *Neural Computation*, 5(3):443-455, 1993.
- [91] N. Intrator. On the combination of supervised and unsupervised learning. *Physical Review A*, 1993.
- [92] N. Intrator. On the use of projection pursuit constraints for training neural networks. In C. L. Giles, S. J. Hanson, and J. D. Cowan, editors, *Advances in Neural Information Processing Systems*, volume 5. Morgan Kaufmann, San Mateo, CA, 1993.
- [93] N. Intrator, M. F. Bear, L N Cooper, and M. A. Paradiso. Theory of synaptic plasticity in visual cortex. In R. Thompson, M. Baudry, and J. Davis, editors, *Synaptic Plasticity: Molecular, Cellular and Functional Aspects*. MIT Press, Cambridge, MA, 1993.
- [94] N. Intrator and J. I. Gold. Three-dimensional object recognition using an unsupervised BCM network: The usefulness of distinguishing features. *Neural Computation*, 5:61-74, 1993.
- [95] Y. Liu. Neural network model selection using asymptotic jackknife estimator and cross-validation method. In D. Touretzky, editor, *Advances Neural Information Processing Systems*, volume 5. Morgan Kaufmann, San Mateo, CA, 1993.

- [96] M. P. Perrone and L. N Cooper. Coulomb potential learning. In *The Handbook of Brain Theory and Neural Networks*. MIT Press, 1993. [To Appear].
- [97] Michael P. Perrone and Leon N Cooper. Learning from what's been learned: Supervised learning in multi-neural network systems. In *Proceedings of the World Conference on Neural Networks*. INNS, 1993. [To appear].
- [98] Michael P. Perrone and Leon N Cooper. When networks disagree: Ensemble method for neural networks. In R. J. Mammone, editor, *Neural Networks for Speech and Image processing*. Chapman-Hall, 1993. [In press].

## TECHNICAL REPORTS

- [1] E. L. Bienenstock, L. N Cooper, and P. W. Munro. Theory for the development of neuron selectivity: Orientation specificity and binocular interaction in visual cortex. Technical report, Brown University, June 1981.
- [2] M. K. Ellis and J. D. Daniels. Catecholamine enhancement and visual cortex plasticity in developing kittens. Technical report, Brown University, March 1982.
- [3] M. F. Bear and J. D. Daniels. The plastic response to monocular deprivation persists after chronic depletion of norepinephrine in kitten visual cortex. Technical report, Brown University, June 1982.
- [4] J. D. Daniels, M. Schwartz, S. A. Bianco, M. K. Ellis, S. B. Nelson, M. F. Bear, and M. Garrett. One small randomly blinking dot in an otherwise dark environment: Effects on visual cortical neurons of kittens. Technical report, Brown University, June 1982.
- [5] J. D. Daniels and T. R. Myers. An automated optical display system for visual physiology experiments. Technical report, Brown University, July 1982.
- [6] L. N Cooper, P. W. Munro, and C. L. Scofield. Neuron selectivity: Single neuron and neuron networks. Technical report, Brown University, July 1982.
- [7] M. A. Paradiso, M. F. Bear, and J. D. Daniels. Effects of intracortical infusion of 6-hydroxydopamine on the response of kitten visual cortex to monocular deprivation. Technical report, Brown University, March 1983.
- [8] P. W. Munro. Neural plasticity: Single models for discrimination and generalization and an experimental ensemble approach. Technical report, Brown University, April 1983.
- [9] A. G. Knapp and J. A. Anderson. A theory of categorization based on distributed memory storage. Technical report, Brown University, October 1983.
- [10] L. N Cooper. Neuron learning to network organization. Technical report, Brown University, December 1983.
- [11] J. D. Daniels, E. Pressman, M. A. Schwartz, S. B. Nelson, and D. J. Kraus. Effects of luminance and flicker on ocular dominance shift in kitten visual cortex. Technical report, Brown University, January 1984.
- [12] A. H. Kawamoto and J. A. Anderson. A neural network model of multistable perception. Technical report, Brown University, February 1984.
- [13] D. Ryan, S. Veillette, and J. D. Daniels. Automatic control of a visual physiology experiment, using minc. Technical report, Brown University, April 1984.
- [14] L. N Cooper. A model for the development of neurons in visual cortex. Technical report, Brown University, July 1984.



- [15] M. A. Paradiso and L. N Cooper. Correlation of afferent activity and binocular receptive field properties. Technical report, Brown University, July 1984.
- [16] C. L. Scofield. The development of selectivity and ocular dominance in a neural network. Technical report, Brown University, October 1984.
- [17] M. A. Paradiso. Experimental and theoretical studies of the constraints on development and plasticity in visual cortex. Technical report, Brown University, October 1984.
- [18] M. F. Bear, K. M. Carnes, and F. F. Ebner. An investigation of cholinergic circuitry in cat striate cortex using acetylcholinesterase histochemistry. Technical report, Brown University, October 1984.
- [19] S. B. Nelson, M. A. Schwartz, and J. D. Daniels. Clonidine and cortical plasticity: Possible evidence for noradrenergic involvement. Technical report, Brown University, October 1984.
- [20] P. W. Munro. A model for generalization and specification by single neurons. Technical report, Brown University, February 1985.
- [21] H. W. Winston. A neural model of a semantic network. Technical report, Brown University, June 1985.
- [22] M. F. Bear, K. M. Carnes, and F. F. Ebner. Postnatal changes in the distribution of acetylcholinesterase in kitten striate cortex. Technical report, Brown University, February 1985.
- [23] L. N Cooper and C. L. Scofield. Recent developments in neural models. Technical report, Brown University, March 1985.
- [24] M. F. Bear. An investigation of the mechanisms and modulators of developmental plasticity in kitten visual cortex. Technical report, Brown University, June 1985.
- [25] F. F. Ebner, M. F. Bear, and D. E. Schmechel. Glutamic acid decarboxylase in kitten striate cortex. Technical report, Brown University, March 1985.
- [26] H. Kucera. Language, its mathematical structure, and computers. Technical report, Brown University, July 1985.
- [27] L. N Cooper. Cortical plasticity: Theoretical analysis, experimental results. Technical report, Brown University, July 1985.
- [28] L. N Cooper. Local and global factors in learning. Technical report, Brown University, November 1985.
- [29] J. P. Donoghue. Peripheral nerve injury in developing rats reorganizes motor cortex. Technical report, Brown University, May 1986.
- [30] J. A. Anderson and G. Murphy. Psychological concepts in a parallel system. Technical report, Brown University, June 1986.
- [31] J. D. Daniels and A. B. Saul. Modeling and simulation i: Introduction and guidelines. Technical report, Brown University, June 1986.

- [32] L. N Cooper. Theory of an immune system retrovirus. Technical report, Brown University, July 1986.
- [33] J. D. Daniels and Krausse. Cortical plasticity as revealed by ocular dominance shift: Effects of limited visual environments. Technical report, Brown University, November 1986.
- [34] A. B. Saul. Visual cortical unit response properties in kittens given brief monocular experience following dark rearing. Technical report, Brown University, December 1986.
- [35] A. B. Saul and J. D. Daniels. Modeling and simulation ii: Specificity models for visual cortex development. Technical report, Brown University, December 1986.
- [36] A. B. Saul and E. E. Clothiaux. Modeling and simulation iii: Simulation of a model for development of visual cortical specificity. Technical report, Brown University, December 1986.
- [37] F. F. Ebner C. F. Hohmann, M. F. Bear. Glutamic acid decarboxylase activity decreases in mouse neocortex after lesions of the basal forebrain. Technical report, Brown University, December 1986.
- [38] L. N Cooper. Future of brain and information research. Technical report, Brown University, May 1987.
- [39] C. M. Bachmann. The hopfield model and beyond. Technical report, Brown University, May 1987.
- [40] A. Kleinschmidt, M. F. Bear, and W. Singer. Evidence for an involvement of NMDA receptor mechanisms in experience-dependent plasticity of kitten striate cortex. Technical report, Brown University, May 1987.
- [41] C. M. Bachmann, L. N Cooper, A. Dembo, and O. Zeitouni. A relaxation model for memory with high storage density. Technical report, Brown University, June 1987.
- [42] A. Dembo and O. Zeitouni. General potential surfaces and neural networks. Technical report, Brown University, June 1987.
- [43] M. F. Bear, L. N Cooper, and F. F. Ebner. The physiological basis of a theory for synapse modification. Technical report, Brown University, June 1987.
- [44] L. N Cooper and C. L. Scofield. Mean field theory of a neural network. Technical report, Brown University, January 1988.
- [45] T. W. Potter. Storing & retrieving data in a parallel distributed memory system. Technical report, Brown University, June 1988.
- [46] J. N. Sanes, S. Suner, and J. P. Donoghue. Adult motor cortex somatic representation patterns reorganize after motor nerve injury. Technical report, Brown University, June 1988.
- [47] M. F. Bear, A. Kleinschmidt, and W. Singer. Experience-dependent modifications of kitten striate cortex are not prevented by thalamic lesions that include the intralaminar nuclei. Technical report, Brown University, January 1989.

- [48] M. F. Bear and L. N Cooper. Molecular mechanisms for synaptic modification in the visual cortex: Interaction between theory and experiment. Technical report, Brown University, February 1989.
- [49] S. M. Dudek, W. D. Bower, and M. F. Bear. Postnatal changes in glutamate stimulated phosphoinositol turnover in rat neocortical synaptoneurosomes. Technical report, Brown University, February 1989.
- [50] D. L. Reilly and L. N Cooper. An overview of neural networks: Early models to real world systems. Technical report, Brown University, June 1989.
- [51] C. M. Bachmann. Gain modification enhances high momentum backward propagation. Technical report, Brown University, December 1989.
- [52] R. L. Neve and M. F. Bear. Visual experience regulates gene expression in the developing visual cortex. Technical report, Brown University, June 1989.
- [53] N. Intrator. A neural network for feature extraction. Technical report, Brown University, April 1990.
- [54] N. Intrator. An averaging result for feature extraction. Technical report, Brown University, April 1990.
- [55] E. Clothiaux, M. Bear, and L. N Cooper. Synaptic plasticity in visual cortex: Comparison of theory with experiment. Technical report, Brown University, May 1991.
- [56] N. Intrator. Feature extraction using an unsupervised neural network. Technical report, Brown University, May 1991.
- [57] N. Intrator and L. N. Cooper. Objective function formulation of the BCM theory of visual cortical plasticity: Statistical connections, stability conditions. Technical report, Brown University, June 1992.
- [58] N. Intrator and G. Tajchman. Supervised and unsupervised feature extraction from a cochlear model for speech recognition. Technical report, Brown University, December 1992.
- [59] M. P. Perrone. A novel recursive partitioning criterion. Technical report, Brown University, December 1992.
- [60] S. M. Dudek and M. F. Bear. Homosynaptic long-term depression in area CA1 of hippocampus and the effects on NMDA receptor blockade. Technical report, Brown University, December 1992.
- [61] M. P. Perrone and L. N Cooper. When networks disagree: Ensemble method for neural networks. Technical report, Brown University, December 1992.
- [62] N. Intrator, M. F. Bear, L N Cooper, and M. A. Paradiso. Theory of synaptic plasticity in visual cortex. Technical report, Brown University, December 1992.

- [63] N. Intrator, J. I. Gold, H. H. Bülthoff, and S. Edelman. 3D Object recognition using an unsupervised neural network: Understanding the distinguishing features. Technical report, Brown University, December 1992.
- [64] Y. Liu. Neural network model selection using asymptotic jackknife estimator and cross-validation method. Technical report, Brown University, May 1993.
- [65] N. Intrator and J. I. Gold. Three-dimensional object recognition using an unsupervised BCM network: The usefulness of distinguishing features. Technical report, Brown University, May 1993.
- [66] M. P. Perrone. A soft-competitive splitting rule for adaptive tree-structured neural networks. Technical report, Brown University, May 1993.
- [67] M. P. Perrone and N. Intrator. Unsupervised splitting rules for neural tree classifiers. Technical report, Brown University, May/June 1993.
- [68] Y. Liu and H. Shouval. Principal components of natural images - an analytical solution. Technical report, Brown University, May 1993.

## ABSTRACTS

- [1] J. D. Daniels, M. K. Ellis, S. A. Bianco, M. Garrett, S. B. Nelson, and M. Schwartz. Catecholamine depletion, ocular dominance shift and direction selectivity in kitten visual cortex. In *Society for Neuroscience*, 1981.
- [2] M. F. Bear, M. A. Paradiso, and J. D. Daniels. Visual cortical plasticity: Deficit after acute, but not chronic, noradrenergic denervation with 6-hydroxydopamine. In *Society for Neuroscience*, 1982.
- [3] P. W. Munro, C. L. Scofield, and L. N. Cooper. A theoretical framework encompassing generalizing and discriminating units applied to feature sensitive neurons in visual cortex. In *Society for Neuroscience*, 1982.
- [4] M. F. Bear, R. J. Clinton, and D. A. Haycock. Stability of 6-hydroxydopamine under minipump conditions. In *Society for Neuroscience*, 1983.
- [5] J. D. Daniels, S. B. Nelson, and M. Schwartz. Restricted visual environments and ocular dominance shifts. In *ARVO*, 1983.
- [6] S. B. Nelson, M. Schwartz, and J. D. Daniels. Clonidine and visual cortical plasticity: New evidence for noradrenergic involvement. In *Society for Neuroscience*, 1983.
- [7] M. A. Paradiso. Theoretical development of binocular receptive fields in cat visual cortex. In *Society for Neuroscience*, 1983.
- [8] M. F. Bear, K. Landay, D. E. Schmechel, and F. F. Ebner. Glutamic acid decarboxylase in the striate cortex of normal and monocularly deprived kittens. In *ARVO*, 1984.
- [9] K. M. Carnes, M. F. Bear, and F. F. Ebner. The cholinergic enervation of cat striate cortex. In *ARVO*, 1984.
- [10] C. Aoki, P. Siekevitz, and J. D. Daniels. Two methods of catecholamine depletion that yield different effects on plasticity both depress ne-stimulation of adenylate cyclase ( $\alpha$ -case) activity. In *Society for Neuroscience*, 1985.
- [11] J. D. Daniels and A. B. Saul. Ocular dominance, selectivity, and responsiveness in kitten area 17 neurons, after dark rearing plus brief monocular experience. In *Society for Neuroscience*, 1985.
- [12] J. P. Donoghue, B. Z. Berger, S. Ryan, and J. Sanes. Reorganization of the somatic representation in motor cortex following forelimb amputation in newborn rats. In *Society for Neuroscience*, 1985.
- [13] A. B. Saul and J. D. Daniels. Adaptation effects from conditioning area 17 cortical units in kittens during physiological recording. In *Society for Neuroscience*, 1985.

- [14] W. Singer and M. F. Bear. 6-hydroxydopamine interferes with cholinergic transmission in striate cortex. In *Society for Neuroscience*, 1985.
- [15] J. D. Daniels, M. Motuz, and A. Pelah. A cosine function predicts the branching angle of an axon bifurcation as a ratio of cost before and after the junction. In *Society for Neuroscience*, 1986.
- [16] J. D. Daniels and A. B. Saul. One week's monocular experience in 16-hz strobe illumination is adequate to promote complete ocular dominance shift in monocularly deprived kittens. In *ARVO Supplement to Investigative Ophthalmology and Visual Science*, 1986.
- [17] T. J. Pincince and J. P. Donoghue. Receptive field architecture in the whisker representation of rat somatosensory cortex. In *Society for Neuroscience*, 1986.
- [18] S. Suner, J. P. Donoghue, B. L. Schlaggar, and J. N. Sanes. Peripheral injury induces motor cortex reorganization in adult rats. In *Society for Neuroscience*, 1986.
- [19] K. Barstad and M. F. Bear. The cholinergic innervation of somatic sensory cortex in the cat. In *Society for Neuroscience*, 1987.
- [20] M. F. Bear, Q. Gu, A. Kleinschmidt, and W. Singer. Effects of intracortical infusion of apv on synaptic modifications in kitten striate cortex. In *Society for Neuroscience*, 1987.
- [21] J. P. Donoghue, S. Suner, J. F. Lando, and J. N. Sanes. Motor cortical representation patterns shift rapidly following motor nerve section. In *Society for Neuroscience*, 1987.
- [22] E. Clothiaux, M. F. Bear, and L. N. Cooper. Experience-dependent synaptic modifications in the visual cortex studied using a neural network model. In *Society for Neuroscience*, 1988.
- [23] J. D. Daniels, L. Sewall, and C. Brennan. Hysteresis, memory & attention in electronic neural networks. In *Society for Neuroscience*, 1988.
- [24] S. M. Dudek, W. D. Bowen, and M. F. Bear. Postnatal changes in glutamate-stimulated phosphatidyl inositol turnover in neocortical synaptoneurosome. In *Society for Neuroscience*, 1988.
- [25] N. Intrator and B. S. Seebach. A learning mechanism for the identification of hidden structures in signal sets. In *International Neural Network Society*, 1988.
- [26] K. Jacobs and J. P. Donoghue. Inhibition shapes the size of motor cortex representations. In *Society for Neuroscience*, 1988.
- [27] M. Sahin, W. D. Bowen, and J. P. Donoghue. Cellular localization of cholinergic and opiate receptors in rat sensorimotor cortex. In *Society for Neuroscience*, 1988.
- [28] B. S. Seebach and N. Intrator. Learning mechanism for the identification of acoustic features. In *Society for Neuroscience*, 1988.
- [29] J. E. Sherin, D. Feldman, and M. F. Bear. NMDA-evoked calcium uptake by slices of kitten visual cortex maintained in vitro. In *Society for Neuroscience*, 1988.

- [30] H. Colman and M. F. Bear. Blockade of visual cortical NMDA receptors prevents the shrinkage of lateral geniculate neurons following monocular deprivation. In *Society for Neuroscience*, 1989.
- [31] S. M. Dudek and M. F. Bear. Ibotenate-stimulated phosphoinositide turnover: a biochemical correlate of the critical period for synaptic plasticity. In *Society for Neuroscience*, 1989.
- [32] N. Intrator. A neural network for feature extraction. In *Proceedings of the Neural Information Processing Systems*, 1989.
- [33] B. S. Seebach and N. Intrator. Acoustic feature development during unsupervised learning by a neural net. In *Acoustical Society*, 1989.
- [34] C. C. Law and L. N Cooper. A network model of the effects of deprivation and pharmacological agents on the response properties of kitten visual cortex. In *Society for Neuroscience*, 1991.
- [35] M. P. Perrone. A novel recursive partitioning criterion. In *Proceedings of the International Joint Conference on Neural Networks*, 1991.
- [36] H. H. Bülthoff, S. Edelman, E. Sklar, and N. Intrator. Image-based features in the recognition of novel 3D objects. In *Investigative Ophthalmology and Visual Science*, 1992.
- [37] L. N Cooper. Synaptic plasticity in the visual cortex: Toward a molecular basis for learning and memory storage (plenary lecture). In *Proceedings of the International Joint Conference on Neural Networks*, 1992.
- [38] G. N. Tajchman and N. Intrator. Feature extraction from a cochlear model representation: A novel supervised/unsupervised neural network hybrid for speech recognition. In *Journal of the Acoustical Society of America*, 1992.

## PH.D. THESES

- [1] Paul W. Munro. *Neural Plasticity: Single Neuron Models for Discrimination and Generalization and an Experimental Ensemble Approach*. PhD thesis, Brown University, Dept. of Physics; Dr. Leon N Cooper, Thesis Supervisor, June 1983.
- [2] Mark F. Bear. *An Introduction to the Mechanisms and Modulators of Developmental Plasticity in the Kitten Visual Cortex*. PhD thesis, Brown University, Dept. of Biology and Medicine; Dr. Ford F. Ebner, Thesis Supervisor, June 1984.
- [3] Michael A. Paradiso. *Experimental and Theoretical Studies of the Constraints on Development and Plasticity in Visual Cortex*. PhD thesis, Brown University, Dr. Leon N Cooper, Thesis Supervisor, June 1984.
- [4] Christopher L. Scofield. *The Development of Selectivity and Ocular Dominance in a Neural Network*. PhD thesis, Brown University, Dept. of Physics; Dr. Leon N Cooper, Thesis Supervisor, June 1984.
- [5] Howard A. Winston. *A Neural Model of a Semantic Network*. PhD thesis, Brown University, Dept. of Physics; Dr. James A. Anderson, Thesis Supervisor, June 1984.
- [6] Alan H. Kawamoto. *Dynamic Processes in the (Re)Solution of Lexical Ambiguity*. PhD thesis, Brown University, Dept. of Psychology; Dr. James A. Anderson, Thesis Supervisor, May 1985.
- [7] Alan B. Saul. *Visual Cortical Unit Response Properties in Kittens Given Brief Monocular Experience Following Dark Rearing*. PhD thesis, Brown University, Dept. of Applied Mathematics; Dr. Leon N Cooper, Thesis Supervisor, May 1986.
- [8] Terry W. Potter. *Storing and Retrieving Data in a Parallel Distributed Memory System*. PhD thesis, Advanced Technology, SUNY at Binghamton; Dr. Leon N Cooper, Thesis Supervisor, May 1987.
- [9] Charles M. Bachmann. *Learning and Generalization in Neural Networks*. PhD thesis, Brown University, Dept. of Physics; Dr. Leon N Cooper, Thesis Supervisor, May 1990.
- [10] Eugene Clothiaux. *Theoretical and Empirical Study of Visual Cortex Using the BCM Neural Network Model*. PhD thesis, Brown University, Dept. of Physics; Dr. Leon N Cooper, Thesis Supervisor, May 1990.
- [11] Nathan Intrator. *Feature Extraction Using an Exploratory Projection Pursuit Neural Network*. PhD thesis, Brown University, Div. of Applied Mathematics; Dr. Leon N Cooper, Thesis Supervisor, May 1990.
- [12] Bradley S. Seebach. *Evidence for the Development of Phonetic Property Detectors in a Neural Net without Innate Knowledge of Linguistic Structure*. PhD thesis, Brown University, Center for Neural Science; Dr. Leon N Cooper, Thesis Supervisor, May 1990.



- [13] Michael P. Perrone. *Improving Regression Estimation: Averaging Methods for Variance Reduction with Extensions to General Convex Measure Optimization*. PhD thesis, Brown University, Institute for Brain and Neural Systems; Dr. Leon N Cooper, Thesis Supervisor, May 1993.

## UNDERGRADUATE HONORS THESES

- [1] Kristen E. Barstad. The cholinergic innervation of somatic sensory cortex in the cat. Center for Neuroscience, May 1988.
- [2] Cameron Brennan. Visual response properties of single units in PMLS and EVA visual areas of the cat. Center for Neuroscience, May 1988.
- [3] Daniel Feldman. Pharmacological modulation of n-methyl-d-aspartic acid induced calcium fluxes in kitten striate cortex. Center for Neuroscience, May 1988.
- [4] Jonathan E. Sherin. The effects of dark rearing on NMDA-stimulated  $45\text{Ca}^{++}$  uptake into area 17 cortical slices in the kitten. Center for Neuroscience, May 1988.
- [5] Howard Colman. The effects of visual cortical NMDA receptor blockade on the shrinkage of LGN neurons following monocular deprivation. Center for Neuroscience, May 1989.
- [6] Mark Weisskopf. Prolonged enhancement of neural transmission in grafted and normal rat si cortex. Center for Neuroscience, May 1989.
- [7] Christian Ivan Fras. The effects of sensory experience on calcium homeostasis in the visual cortex. Center for Neuroscience, May 1990.
- [8] William Press. Long-term potentiation in kitten striate cortex. Center for Neuroscience, May 1990.
- [9] Joel Gold. Long-term depression in rat hippocampal slices. Center for Neuroscience, May 1991.
- [10] Josh Gold. A biophysical model of spine calcium yielding insights into mechanisms of a theory of synaptic plasticity. Center for Neuroscience, May 1991.
- [11] David Stellwagen. The effect of activity on plasticity and calcium buffering in the cerebral cortex. Center for Neuroscience, May 1992.
- [12] Manish Butte. Neural network techniques for time series prediction. Institute for Brain and Neural Systems, May 1993.